



Differential Diagnosis of Paradoxical Vocal Fold Movement

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Purpose: To present the differential diagnosis of paradoxical vocal fold movement (PVFM) and its distinguishing features.

Methods: The authors provide an overview of PVFM by drawing from 30 years of clinical and research experience, and relating that experience to literature in this area.

Conclusion: PVFM is characterized by inappropriate adduction of the vocal folds during inspiration. PVFM is an uncommon and sometimes confusing cause of airway obstruction.

The resultant obstruction may be intermittent or continuous, mild or severe. Most patients with PVFM have a specific etiology— inflammatory, neurological, neoplastic, iatrogenic, or psychological—that influences type of treatment and outcome.

Key Words: stridor, paradoxical vocal fold movement, paradoxical vocal cord movement, vocal cord dysfunction

During the respiratory cycle of higher animals and human beings, the vocal folds partially abduct with inhalation and partially adduct with exhalation (Ward, Hanson, & Berci, 1981). This phasic vocal fold movement is physiological and allows the unimpeded movement of air into the lungs during inspiration while helping to maintain the alveolar patency of the lungs by providing positive airway pressure during expiration.

Some patients who present with dyspnea, stridor, and airway obstruction have paradoxical vocal fold movement (PVFM). PVFM is characterized by inappropriate adduction of the vocal folds during inspiration (Appleblatt & Baker, 1981; Maschka et al., 1997; Mathers-Schmidt, 2001; Murry, Tabae, & Aviv, 2004). The persistence and the degree of inappropriate glottal closure with PVFM determines the degree of obstruction. In some patients, the problem is constant and severe, requiring airway intervention; in others, the problem is intermittent and relatively mild.

In addition to the term PVFM, there have been a number of other terms used in the literature to identify this complex and often confusing disorder in adults and children, including Munchausen's stridor (Patterson, Schatz, & Horton, 1974), vocal cord dysfunction (Christopher et al., 1983), paradoxical vocal cord motion (Martin, Blager, Gay, & Wood, 1987; Sandage & Zelazny, 2004), and irritable larynx syndrome (Andrianopoulos, Gallivan, & Gallivan, 2000; Morrison, Rammage, & Emami, 1999). Maschka et al. (1997)

and Mathers-Schmidt (2001) provided classifications and discussions of some of the causes of this condition.

In this article, we provide a concise differential diagnosis for PVFM in adults (see Appendix A). We reserve the term PVFM to refer to inappropriate vocal fold adduction during inspiration, and we present differentiating features of specific etiologies. We offer a new classification of PVFM because accurate diagnosis is a prerequisite for effective treatment.

Differential Diagnosis of PVFM

Reactive upper airway obstruction virtually always occurs at the laryngeal level, while asthma is the most common reactive lower airway disease. Sometimes tracheal diseases, such as tracheomalacia, intrinsic or extrinsic neoplastic laryngotracheal growths (e.g., subglottic stenoses, carcinoid tumors, thyroid tumors), and bronchial/pulmonary diseases (e.g., asthma, chronic obstructive pulmonary disease), may be mistaken for reactive upper airway disease (Altman et al., 2002; Brugman & Simons, 1998).

The pattern of stridor (noisy breathing) is the key differentiating factor between upper and lower airway obstruction (see Appendix B). Stridor can be inspiratory, expiratory, or both (biphasic). In the authors' experience, most PVFM patients present with stridor. There are some patients who do not have stridor but rather, mild airway obstruction. PVFM

that is triggered by exercise, for example, may or may not be associated with stridor.

Patients with fixed mechanical problems, such as tracheal tumors, have signs and symptoms that are slowly progressive and usually relatively self-evident. For example, if a patient has thyroid cancer and inspiratory stridor, the laryngeal examination may show bilateral vocal fold paralysis and the presence of a subglottic mass. Progressive airway obstruction is specifically *not* usually characteristic of PVFM. It almost always has a nonprogressive pattern, that is, fluctuation in symptom severity or attacks. If a clinician is seeing a patient with progressive airway obstruction, that pattern is a “red flag” that is potentially more ominous and certainly requires a thorough medical examination.

Patients with PVFM often describe “choking” episodes during inspiration, with a sensation of tightness in the throat more than in the chest. But the term “choking” may be used by patients to describe different symptoms: (a) acute-onset breathing problems, such as stridor; (b) dysphagia, such as food getting stuck in the hypopharynx or esophagus; and (c) aspiration (e.g., saliva, liquid, food, or some other foreign body getting into the airway) stimulating violent coughing. Once distinguishing that the patient’s type of “choking” is stridor (such as that associated with PVFM), the clinician is challenged to determine its etiology. Potential etiologies include laryngopharyngeal reflux, respiratory-type laryngeal dystonia, asthma and hyper-immune disorders, brainstem abnormalities, drug-induced (laryngeal) dystonic reactions, and psychogenic stridor.

Laryngopharyngeal Reflux

Laryngopharyngeal reflux (LPR) is the most common cause of PVFM in the authors’ experience (Koufman, 1991; Little et al., 1997). Reflux-related PVFM is usually intermittent, typically lasting minutes to hours (sometimes days), and does not usually require airway intervention. The respiratory obstruction seen with PVFM is believed to be related to stimulation of vagally mediated reflexes by exposure of gastric fluids to supraglottic chemoreceptors (Loughlin et al., 1996). It has been shown in an animal model that the afferent limb of the PVFM reflex is innervated by the superior laryngeal nerves and that the efferents are innervated by the recurrent laryngeal nerves (Duke et al., 2001; Loughlin et al., 1996).

Patients with LPR-related PVFM usually have other clues that they have LPR. Often the attacks follow a pattern, such as occurring after a meal, after the start of exercise, or after bending over. Sometimes the attacks may wake the patient from a sound sleep. Other times, the attacks may occur without any pattern or identifiable precipitant, but the patient may have symptoms and laryngeal findings of LPR (Little et al., 1997). Some patients have several attacks each day, while others have as few as one or so a year.

LPR is often silent, occurring without heartburn. Only about 30%–40% of patients with LPR report ever having heartburn (Koufman, 1991). However, patients with LPR do complain of symptoms such as chronic or intermittent hoarseness, difficulty swallowing, a globus sensation (i.e., lump in the throat), chronic throat clearing, cough, and postnasal drip.

While not the focus of this article, it is important to recognize that paroxysmal laryngospasm (LS) is also often caused by LPR, and that it is sometimes confused with PVFM. Loughlin and Koufman (1996) reported clinical data from 15 consecutive patients with LS, 12 of whom underwent ambulatory, 24-hr, double-probe (simultaneous esophageal and pharyngeal) pH monitoring. Almost all (11/12) had pH-documented LPR. All of the patients were treated with dietary and lifestyle modification as well as twice-daily proton pump inhibitors. Within 4 weeks, all attacks had ceased in all 15 patients.

LPR-related LS is more common than PVFM. Although they both operate under the same neurogenic mechanism, there are important clinical differences. The single most important difference is that during LS, the vocal folds simply do not abduct. In contrast, during PVFM, there is vocal fold *movement*, albeit reduced and paradoxical. See Table 1 for differentiating features between LS and PVFM.

Respiratory-Type Laryngeal Dystonia

After LPR, respiratory-type laryngeal dystonia is the second most common cause of PVFM (in the authors’ experience), even though it is infrequently reported in the medical literature (Blitzer & Brin, 1991; Grillone, Blitzer, Brin, Annino, & Saint-Hilaire, 1994; Marion, Klap, & Cohen, 1992). The senior author has seen approximately 30 cases in 30 years of laryngology practice. Respiratory-type laryngeal dystonia and spasmodic dysphonia are both

TABLE 1. Typical differentiating features of laryngospasm (LS) and paradoxical vocal fold movement (PVFM).

Feature	LS	PVFM
History and symptoms		
Duration of attacks	Minutes	Minutes-hours
Sudden (acute) onset	Yes	Sometimes
Stridor at rest during attacks	Yes	Sometimes
Associated cough and hoarseness	Usually	Sometimes
Laryngeal examination		
Findings of laryngopharyngeal reflux	Yes	Sometimes
Adductions during inspiration	No	Usually
Brisk abduction with sniffing	Yes	Yes
Failure of any abduction during attacks	Yes	No

focal laryngeal dystonias, although patients rarely have both conditions. Both may affect breathing, but the manifestations are different. With respiratory-type laryngeal dystonia, patients experience PVFM, whereas with spasmodic dysphonia, usually just speech-breathing is affected.

The diagnosis of respiratory-type dystonia depends on the medical history and laryngeal findings. Usually the history will reveal that the patient has continual but varying degrees of breathing problems during the day but not at night, during sleep. This type of daytime PVFM is variably task-specific. In other words, during transnasal flexible laryngoscopy, the examiner will usually observe a degree of PVFM. Patients with a neurogenic etiology are expected to have more continuous symptoms than patients with other PVFM causes. However, during transnasal flexible laryngoscopy, when the patient is asked to take a deep breath, the paradoxical adductions worsen noticeably.

While respiratory-type laryngeal dystonia can be confused with the other causes of PVFM, the important features of this diagnosis are that the patient reports relative airway obstruction all day, every day, that PVFM is observed during the laryngeal examination, and that the signs of PVFM worsen with the deep-breath task. Finally, these patients usually respond dramatically to unilateral vocal fold injections of 0.5–2.5 units of botulinum toxin A, with many months of relief.

Asthma and Hyper-Immune Disorders

It has been suggested that PVFM can occur with asthma as much as 40%–50% of the time (Newman, Mason, & Schmaling, 1995); however, that is not the authors' experience. Indeed, we have found that patients with PVFM have often been misdiagnosed as having asthma as the cause of their airway obstruction, despite the fact that antiasthma medications provide no relief.

There does appear to be a small subset of patients who have both asthma and PVFM (Collett, Brancatisano, & Konno, 1983; Harding & Richter, 1997; Martin et al., 1987; Perkner et al., 1998). Although the mechanism of PVFM in these patients is unknown, glottal aperture changes have been observed in patients with asthma (Collett et al., 1983), and vagal reflex dysfunction may be the cause. When inspiratory stridor is present in any patient with asthma, the diagnosis of PVFM should be considered. The finding of PVFM on transnasal flexible laryngoscopy is diagnostic.

Allergens, chemicals, and inhaled irritants have been reported to cause PVFM (Perkner et al., 1998). Hypersensitivity reactions should be differentiated from true allergic responses, even though the pathophysiology of such reactions is difficult to explain. Among the commonly identified precipitants are temperature change (commonly cold) and volatile aromatics such as perfumes. While allergens and chemicals can be related to reactive airway disease, many patients with such environmental triggers are found to be positive for LPR with pH testing. The relationships between reflux, reactive airway diseases, and environmental precipitants need systematic study.

Brainstem Abnormalities

Central neurological brainstem abnormalities can cause PVFM, in addition to or complicated by bilateral abductor paralysis, apneic episodes, and/or central sleep apnea syndrome (Charney, Rorke, Sutton, & Schut, 1987; Holinger, Holinger, Reichert, & Holinger, 1978). Severe closed head injury, Chiari malformations I and II, meningomyelocele, and cerebrovascular accidents (strokes of the posterior circulation) may all produce PVFM. In these cases, the obstruction and inappropriate vocal fold movement are fairly consistent from examination to examination. In addition, the stridor does not disappear during sleep. In addition, patients with PVFM due to brainstem disease often have other neurological problems, and they frequently have sufficiently severe airway obstruction to require airway intervention (Holinger et al., 1978).

In some cases, days or weeks after closed head injury, extubation may fail or the patient may require multiple intubations and then later tracheotomy. With time, as the cerebral and brainstem edema subsides, vocal fold movement may return to normal and the stridor may disappear. Similarly, some patients with congenital lesions of the brainstem may experience a normalization of vocal fold function after surgical treatment of the lesion (Charney et al., 1987).

Drug-Induced (Laryngeal) Dystonic Reactions

Temporary drug-induced PVFM has been reported after administration of neuroleptic drugs such as phenothiazines, including chlorpromazine and haloperidol (Koek & Pi, 1989). This cause of PVFM should be considered in patients receiving such medications. This type of drug-induced stridor is usually associated with extrapyramidal signs and symptoms, including muscle stiffness, and movement disorders of the head and neck such as torticollis. The airway obstruction (PVFM) and the other symptoms are relatively short-lived (minutes to hours) and are reversible with intravenous administration of anticholinergic drugs (Stoelting, 1987).

PVFM may also be seen in association with emergence from general anesthesia, especially after surgery. It appears to be associated with phenothiazine and thiopental use (Stoelting, 1987). Anesthesia-related, drug-induced PVFM may explain some cases of prolonged LS observed during emergence from anesthesia. It has been shown experimentally that such LS can be prevented by the administration of topical lidocaine (Koufman, 1993).

Psychogenic Stridor

Many PVFM patients were referred to us with the diagnosis of psychogenic stridor, but in our experience psychogenic PVFM is very uncommon. Typically, with psychogenic PVFM, respiratory symptoms occur with sudden onset and offset. In addition, such patients are often relatively unconcerned (blasé) about their noisy breathing and airway obstruction. Usually, they have a psychiatric history or obvious secondary gain (Snyder & Weiss, 1989). Some may even casually ask to have a tracheotomy performed.

Psychogenic PVFM patients have inconsistent laryngeal findings on transnasal flexible laryngoscopy, and sometimes the clinician can fool the patient during the examination, making the PVFM go away. Some patients will display bizarre laryngeal behaviors in an attempt to consistently maintain the stridor during sniffing (which usually causes reflexive vocal fold hyperabduction). If the patient is asked to read a long passage aloud, the stridor may disappear and phasic respiratory activity of the vocal folds may normalize.

We believe that psychogenic stridor is overdiagnosed. Patients with presumed psychogenic PVFM should be evaluated by an otolaryngologist, speech-language pathologist, and psychologist or psychiatrist as a team. Other causes of PVFM should be systematically ruled out before “psychogenic” is the diagnosis.

Diagnostic Approach to the Patient With PVFM

The medical history of a patient with difficulty breathing, stridor, or choking episodes should be meticulously obtained until the clinician thoroughly understands the pattern and characteristics of the problem. We provide a clinical worksheet (Appendix C) and a listing of differentiating features of PVFM (see Table 2) to guide the clinician when taking the history, doing a clinical voice evaluation, and doing a laryngeal examination when possible. The laryngeal examination method of choice is transnasal flexible laryngoscopy. *Per oral* examination methods, especially when the tongue of the patient is grasped in a way that alters laryngeal biomechanics, are less useful in the diagnosis of PVFM.

PVFM should be considered when there are findings of (a) inappropriate vocal fold adduction during inspiration (>50% medialization but less than full closure), (b) paroxysmal inability to abduct the vocal folds (also known as transient abductor paralysis), or (c) a combination of those findings. The laryngeal findings of PVFM that have been reported in the literature also include a posterior glottal chink (gap) during inspiration (Christopher et al., 1983) and expiration (Brugman & Simons, 1998; Martin et al., 1987), and when symptomatic and asymptomatic (Treole, Trudeau, & Forrest, 1999).

As part of the workup for the patient suspected of PVFM, we recommend pulmonary function testing, especially spirometry with an inspiratory-expiratory, flow-volume loop. Generally, this test rules in the diagnosis of PVFM and rules out asthma or other lower respiratory diseases. A flat, truncated inspiratory limb of the flow-volume loop is typically

observed in patients with PVFM, but patients with asthma usually show compromised expiratory flow. With PVFM, Murry et al. (2004) reported a reduced ratio of forced inspiratory volume to forced inspiratory vital capacity for patients with PVFM.

Special diagnostic testing may be warranted depending on the suspected cause of PVFM, including serial laryngeal examinations by transnasal flexible laryngoscopy, pH monitoring, neuropsychiatric evaluation, acoustic voice analysis, audio recordings of the patient’s breathing during sleep, and in selected cases, radiographic examinations to rule out neoplastic or structural abnormalities.

LPR is a common cause of PVFM, and we have found that the *reflux symptom index* is a useful clinical parameter (Belafsky, Postma, & Koufman, 2002). This provides a quick and reliable, nine-item, self-reported LPR symptom index. For reflux-induced PVFM, a firm diagnosis can be made when positive pH-monitoring data are combined with the history and laryngeal findings of LPR. In addition, resolution of symptoms with a therapeutic trial of aggressive antireflux therapy confirms the diagnosis.

The medical history gives important clues for patients suspected of having respiratory-type laryngeal dystonia. Unlike patients in the other PVFM groups, patients with respiratory-type laryngeal dystonia describe a progressive onset of the PVFM over a period of weeks, months, or years. In addition, such patients usually deny having other symptoms. On occasion, differentiating dystonic from psychogenic PVFM can be difficult.

Except for patients in the brainstem abnormality group who may require tracheotomy, patients with PVFM usually do not have severe enough respiratory obstruction to warrant airway intervention. Of the other groups of patients with PVFM, patients with dystonia appear next in terms of the frequency of need for airway support, followed by the psychogenic group, the asthma group, and the LPR group. Regardless of the underlying cause, on their first visit, patients with PVFM require counseling and training (breathing recovery exercises) by a speech-language pathologist (Mathers-Schmidt, 2001; Murry et al., 2004; Murry, Tabae, Owczarzak, & Aviv, 2006).

At the time of diagnosis, the authors recommend teaching patients with PVFM the “quick-sniff/slow-blow technique” because it aborts PVFM attacks (Lowry, 2007). This technique involves asking the patient to “sniff in through your nose quickly, and then blow out slowly through your mouth. Purse your lips to hear the air flow out.” Sniffing with flaring of the nostrils is linked with brisk vocal fold abduction and is a brainstem reflex. Teaching this procedure is part of the

TABLE 2. Key differentiating features of the causes of PVFM.

Cause	Pattern	Duration	Dysphonia	Airway support
Reflux	Paroxysmal	Minutes-hours	Usually	Almost never
Dystonia	Daytime	Hours	Rarely	Sometimes
Asthma/allergy	Paroxysmal	Hours-days	Rarely	Sometimes
Brainstem	Continual	Continual	Sometimes	Usually
Drug induced	Continual	Hours-days	Rarely	Sometimes
Psychogenic	Paroxysmal	Variable	Never	Sometimes

process of providing reassurance for patients who are fearful and initiates therapy that may include respiratory exercises that focus on exhalation, rhythmic abdominal movement patterns, resonant voice, and easy-onset voice exercises to reduce associated hyperfunctional voice behaviors.

With PVFM, once a precise etiological diagnosis is suspected or determined, the clinician can make specific treatment recommendations and referrals. Having an accurate diagnosis fosters appropriate expectations for therapy and accelerates its implementation. The variety of techniques and duration of therapy should be individualized according to the underlying cause of PVFM as well as the needs of the patient.

Summary

PVFM presents the clinician with a challenging differential diagnosis. In the authors' experience, the most common causes of PVFM in order of frequency are (a) LPR, (b) respiratory-type laryngeal dystonia, (c) asthma or hyper-immunity-associated PVFM, (d) brainstem abnormalities, (e) drug-induced dystonic reactions, and (f) psychogenic stridor. The workup of PVFM requires a multidisciplinary approach and a variety of diagnostic methods. Likewise, treatment must be individualized.

References

- Altman, K. W., Simpson, C. B., Amin, M. R., Abaza, M., Balkissoon, R., & Casiano, R. (2002). Cough and paradoxical vocal fold motion. *Otolaryngology Head and Neck Surgery, 127*, 501–511.
- Andrianopoulos, M. V., Gallivan, G. J., & Gallivan, K. H. (2000). PVM, PVCD, EPL, and irritable larynx syndrome: What are we talking about and how do we treat it? *Journal of Voice, 14*, 607–618.
- Appleblatt, K. L., & Baker, S. R. (1981). Functional airway obstruction: A new syndrome. *Archives of Otolaryngology, 107*, 305–307.
- Belafsky, P. C., Postma, G. N., & Koufman, J. A. (2002). Validity and reliability of the reflux symptom index (RSI). *Journal of Voice, 16*, 274–277.
- Blitzer, A., & Brin, M. F. (1991). Laryngeal dystonia: A series with botulinum toxin therapy. *Annals of Otolaryngology, Rhinology and Laryngology, 100*, 85–89.
- Brugman, S. M., & Simons, S. M. (1998). Vocal cord dysfunction: Don't mistake it for asthma. *The Physician and Sports Medicine, 26*, 63–74.
- Charney, E. B., Rorke, L. B., Sutton, L. N., & Schut, L. (1987). Management of Chiari II complications in infants with meningomyelocele. *Journal of Pediatrics, 111*, 364–371.
- Christopher, K. L., Wood, R. P., Eckert, R. C., Blager, F. B., Raney, R. A., & Souhrada, J. F. (1983). Vocal-cord dysfunction presenting as asthma. *New England Journal of Medicine, 308*, 1566–1570.
- Collett, P. W., Brancatisano, T., & Konno, K. (1983). Changes in glottic aperture during bronchial asthma. *American Review of Respiratory Diseases, 128*, 719–723.
- Duke, S. G., Postma, G. N., McGuirt, W. F., Jr., Ririe, D., Averill, D., & Koufman, J. (2001). Laryngospasm and diaphragmatic arrest in the immature canine after laryngeal acid exposure: A possible model for sudden infant death syndrome (SIDS). *Annals of Otolaryngology, Rhinology and Laryngology, 110*, 729–733.
- Grillone, G. A., Blitzer, A., Brin, M. F., Annino, D. J., & Saint-Hilaire, M. H. (1994). Treatment of adductor laryngeal breathing dystonia with botulinum toxin type A. *Laryngoscope, 104*, 30–32.
- Harding, S. M., & Richter, J. E. (1997). The role of gastroesophageal reflux in chronic cough and asthma. *Chest, 111*, 1389–1402.
- Holinger, P. C., Holinger, L. D., Reichert, T. J., & Holinger, P. H. (1978). Respiratory obstruction and apnea in infants with bilateral abductor vocal fold paralysis, meningomyelocele, hydrocephalus, and Arnold-Chiari malformation. *Journal of Pediatrics, 92*, 368–373.
- Koek, R. J., & Pi, E. H. (1989). Acute laryngeal dystonic reactions to neuroleptics. *Psychosomatics, 30*, 359–364.
- Koufman, J. A. (1991). The otolaryngologic manifestations of gastroesophageal reflux disease (GERD): A clinical investigation of 225 patients using ambulatory 24-hour pH monitoring and an experimental investigation of the role of acid and pepsin in the development of laryngeal injury. *Laryngoscope, 101*(53), 1–78.
- Koufman, J. A. (1993). Unpublished data.
- Little, J. P., Matthews, B. L., Glock, M. S., Koufman, J. A., Reboussin, D. M., Loughlin, C. J., & McGuirt, W. F., Jr. (1997). Extraesophageal pediatric reflux: 24-hour double-probe pH monitoring of 222 children. *Annals of Otolaryngology, Rhinology and Laryngology, 106*, 1–16.
- Loughlin, C. J., & Koufman, J. A. (1996). Paroxysmal laryngospasm secondary to gastroesophageal reflux. *Laryngoscope, 106*, 1502–1505.
- Loughlin, C. J., Koufman, J. A., Averill, D. B., Cummins, M. M., Kim, Y. J., Little, J. P., et al. (1996). Acid-induced laryngospasm in a canine model. *Laryngoscope, 106*, 1506–1509.
- Lowry, F. (2007, June). *PVFM, chronic cough, throat clearing, hiccups, and aerophagia*. Paper presented at The Voice Foundation's 36th Annual Symposium, Philadelphia.
- Marion, M. H., Klap, P., & Cohen, M. (1992). Stridor and laryngeal focal dystonia. *Lancet, 339*, 457–458.
- Martin, R. J., Blager, F. B., Gay, M. L., & Wood, R. P. (1987). Paradoxical vocal cord motion in presumed asthmatics. *Seminars in Respiratory Critical Care Medicine, 8*, 332–338.
- Maschka, D. A., Bauman, N. M., McCray, P. B., Jr., Hoffman, H. T., Karnell, M., & Smith, R. (1997). A classification scheme for paradoxical vocal cord motion. *Laryngoscope, 107*, 1429–1435.
- Mathers-Schmidt, B. A. (2001). Paradoxical vocal fold motion: A tutorial on a complex disorder and the speech-language pathologist's role. *American Journal of Speech-Language Pathology, 10*, 111–125.
- Morrison, M., Rammage, L., & Emami, A. (1999). The irritable larynx syndrome. *Journal of Voice, 13*, 447–455.
- Murry, T., Tabae, A., & Aviv, J. (2004). Respiratory retraining of refractory cough and laryngopharyngeal reflux in patients with paradoxical vocal fold movement disorder. *Laryngoscope, 114*, 1341–1344.
- Murry, T., Tabae, A., Owczarzak, V., & Aviv, J. (2006). Respiratory retraining therapy and management of laryngopharyngeal reflux in the treatment of patients with cough and paradoxical vocal fold movement. *Annals of Otolaryngology, Rhinology and Laryngology, 115*, 754–758.
- Newman, K., Mason, U., & Schmalig, K. (1995). Clinical features of vocal cord dysfunction. *American Journal of Respiratory Critical Care Medicine, 152*, 1382–1386.
- Patterson, R., Schatz, M., & Horton, M. (1974). Munchausen's stridor: Non-organic laryngeal obstruction. *Clinical Allergy, 4*, 307–310.
- Perkner, J., Fennelley, K., Balkissoon, R., Bartelson, B., Rutenber, A., Wood, R., et al. (1998). Irritant-associated

vocal cord dysfunction. *Journal of Occupational and Environmental Medicine*, 40, 136–143.

Sandage, M., & Zelazny, S. (2004). Paradoxical vocal fold motion in children and adolescents. *Language, Speech, and Hearing Services in Schools*, 35, 353–362.

Snyder, H. S., & Weiss, E. (1989). Hysterical stridor: A benign cause of upper airway obstruction. *Annals of Emergency of Medicine*, 18, 991–994.

Stoelting, R. K. (1987). *Pharmacology and physiology in anesthetic practice*. Philadelphia: Lippincott.

Treole, K., Trudeau, M. D., & Forrest, L. A. (1999). Endoscopic and stroboscopic description of adults with paradoxical vocal fold dysfunction. *Journal of Voice*, 13, 143–152.

Ward, P. H., Hanson, D. G., & Berci, G. (1981). Observations on central neurologic etiology for laryngeal dysfunction. *Annals of Otolaryngology, Rhinology and Laryngology*, 90, 430–441.

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Appendix A

Differential Diagnosis of Paradoxical Vocal Fold Movement in Decreasing Order of Frequency of Occurrence (Authors' Practice)

- Laryngopharyngeal reflux (LPR)
- Respiratory-type laryngeal dystonia
- Asthma-associated and hyper-immune laryngeal dysfunction
- Brainstem abnormalities, such as Chiari malformations
- Drug-induced dystonic reactions
- Psychogenic stridor

Appendix B

Characterization, Localization, and Common Causes of Stridor (Noisy Breathing)

Inspiratory Stridor (Usually Laryngeal)

Bilateral (medial position) vocal fold paralysis
Swelling/edema due to fungal, bacterial, or viral infection
Swelling/edema due to inflammation (e.g., LPR, angioedema)
Obstructing benign growths (e.g., papillomas, cysts, granulomas)
Obstructing (supraglottic, glottic, or infraglottic) laryngeal cancer
Glottic stenosis (e.g., subglottic stenosis, webbing)
Paradoxical vocal fold movement (PVFM)

Inspiratory and Expiratory (Biphasic) Stridor (Usually Tracheal)

Extrinsic compression (e.g., neck masses such as thyroid carcinoma)
Mediastinal compression (e.g., thymic tumors, esophageal or lung cancer)
Laryngeal obstruction without tracheal involvement
PVFM with asthma

Expiratory Stridor (Wheezing; Usually Intrathoracic)

Asthma
Chronic obstructive pulmonary disease (COPD)
Benign and malignant tumors at or near the thoracic inlet

Appendix C (p. 1 of 2)

Voice Institute of New York PVFM Clinical Worksheet

The following worksheet includes a checklist for use when taking a case history and doing a clinical examination. The most important differentiating features of the various causes of PVFM are found in the history. The examination may or may not be confirmatory, because PVFM is often paroxysmal. In other words, careful history taking may help the clinician determine a specific etiology. In the history section below, there are numbers to the right of each item that match the diagnostic possibilities shown:

- L. Laryngopharyngeal reflux (LPR)
- R. Respiratory-type laryngeal dystonia
- A. Asthma-associated & hyper-immune laryngeal dysfunction
- B. Brainstem abnormalities
- D. Drug-induced laryngeal dystonic reactions

We recommend checking each item that applies to the patient, and then adding up the numbers for each corresponding letter where an item is checked. Then, rank the diagnostic groups from high to low by the sums. This may lead the clinician to favor certain diagnoses. Note: This scoring system is intended to be a guide, an adjunct for the clinician. It does not provide a foolproof diagnosis. We have not included psychogenic stridor because its manifestations are so varied. It is a diagnosis of exclusion.

PATIENT HISTORY

	IF POSITIVE - CHECK HERE	L	R	A	B	D
1. Type of Stridor/Breathing Difficulty						
Inspiratory	_____	1	1	1	1	1
Expiratory	_____	0	0	1	1	0
Biphasic (inspiratory & expiratory)	_____	0	0	1	1	1
2. Pattern of Stridor/Breathing Difficulty						
Continuous (all of the time) day & night	_____	0	0	1	1	1
Continuous daytime only not at night	_____	0	1	0	0	0
Intermittent attacks lasting minutes-to-hours	_____	1	0	0	0	1
Intermittent attacks lasting hours-to-days	_____	1	0	1	1	1
Intermittent attacks lasting several days	_____	0	0	0	1	0
3. Triggers (Timing and/or Associated Activities)						
After meals (eating/drinking)	_____	1	0	1	0	0
Awakens from sleep	_____	1	0	1	0	0
Associated with exercise	_____	1	0	1	0	0
Associated with stress	_____	1	1	1	0	0
Associated with certain odors	_____	1	0	1	0	0
4. Associated Symptoms						
Hoarseness	_____	1	1	1	1	1
Chest tightness	_____	1	0	1	0	0
Cough	_____	1	0	1	0	1
Dysphagia	_____	1	0	0	0	0
Globus sensation	_____	1	0	0	0	0
Heartburn	_____	1	0	0	0	0
Regurgitation	_____	1	0	0	0	0
Throat tightness	_____	1	1	1	0	1
5. Specific Relevant Past Medical History						
Allergies and/or asthma	_____	0	0	1	0	1
Brain tumor	_____	0	1	0	1	0
Haldol or other phenothiazine	_____	0	0	0	0	1
Head injury	_____	0	1	0	1	0
Laryngeal or non-laryngeal dystonia	_____	0	1	0	0	0
LPR and/or GERD	_____	1	0	0	0	0
Psychiatric disorder	_____	1	1	1	1	1
Stroke	_____	0	1	0	1	0
Vocal fold paralysis	_____	0	0	0	1	0

Summary Scores

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Voice Institute of New York PVFM Clinical Worksheet

CLINICAL EXAMINATION

IF POSITIVE - CHECK HERE

General Examination/Observations

- Breathy and/or hoarse vocal quality _____
- Inspiratory/biphasic stridor during respiration/speech _____
- Reduced breath support or control _____
- Musculoskeletal tension of the head and neck _____
- Throat tightness/choking/breathing problem during ...
- Alternating /i/ - sniff _____
- Rapid in-and-out breathing _____
- Cough/throat clear/chuckle, then deep breath _____
- Rapid and loud counting _____
- Smelling a strong perfume or chemical _____
- Running in place _____

Laryngeal Examination

- Adduction of vocal folds during ...
- Alternating /i/ - sniff _____
- Rapid in-and-out breathing _____
- Cough/throat clear/chuckle, then deep breath _____
- Inspiratory/biphasic stridor during respiration/speech _____
- Supraglottic muscle tension patterns _____
- Signs of reflux laryngitis _____

Clinical Impressions _____

Recommended Referral(s)

- | | |
|-----------------------------|----------------------------|
| Acoustic analysis _____ | Aerodynamic measures _____ |
| Neurology _____ | Otolaryngology _____ |
| Psychiatry/psychology _____ | Pulmonology _____ |
| Swallow evaluation _____ | Sleep study _____ |